urine microscopy. Four months before admission her haemoglobin concentration had been 13·5 g/dl, erythrocyte sedimentation rate 28 mm in first hour, and serum creatinine concentration 84 μmol/l (0·9 mg/100 ml) and she had been normotensive. Ultrasonography carried out because of abdominal pain showed bilateral hydronephrosis. Intravenous urography showed obstruction on both sides at the same level (L4). Retroperitoneal fibrosis was diagnosed and confirmed at operation. The fibrous tissue zone was 3-4 cm long on both sides. The obstructed ureters were liberated from the fibrous retroperitoneal tissue and transplanted to an intraperitoneal position. Biopsy specimens taken during the operation showed dense collagenous fibrotic tissue typical of retroperitoneal fibrosis. Postoperative recovery was uneventful. Sotalol was stopped and she was discharged well. Five months later she had no symptoms, was normotensive, and had a haemoglobin concentration of 13·7 g/dl and an erythrocyte sedimentation rate of 33 mm in first hour.

Comment

Retroperitoneal fibrosis has been described in association with beta-blockers including atenolol, oxprenolol, amd metoprolol. So far as we know, our patient is the first report of an association with sotalol. Our patient had also been taking a derivative of vitamin A, but no cases of retroperitoneal fibrosis have been reported in association with this drug. No other cause for the retroperitoneal fibrosis was found in our patient. As Thompson and Julian[5] reminded when reporting the association between beta-blockers and retroperitoneal fibrosis, however, we cannot exclude the possibility of an idiopathic case, since beta-blockers are so widely used.

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Chloramine-induced pneumonitis from mixing household cleaning agents

Housewives often mix domestic cleaning agents without being aware of the dangers. For example, mixtures may liberate substantial amounts of irritant gases. We describe a case of severe pneumonitis caused by chloramine released from a mixture of ammonia and household bleach containing sodium hypochlorite.

Case report

A 27-year-old woman mixed about 500 ml of 4·5% household ammonia with the same amount of 5% sodium hypochlorite bleach in a small, poorly ventilated bathroom. Harsh fumes evolved immediately, causing burning in her eyes and throat. Within a few seconds she started choking and coughing violently and later felt nauseated and vomited. Twelve hours after exposure, while still dyspnoeic and coughing, she was admitted to hospital.

On admission the patient was dyspnoeic and tachypnoeic (32 respirations/min). Pulse rate was 130/min and blood pressure was normal. Rales and wheezing were heard throughout both lungs. Chest radiography was unhelpful. Blood gas analysis showed hypoxaemia with oxygen pressure 6·7 kPa (50 mm Hg). Humidified 70% oxygen, terbutaline sulphate inhalations, and intravenous methylprednisolone 1 g thrice daily were administered. Twenty-four hours after exposure she was still tachypnoeic, with no improvement in blood gas values. She was therefore transferred to the intensive care unit and continuous positive airway pressure ventilation was instituted. Pulmonary function values 48 hours after exposure showed a severe restrictive and obstructive pattern with a distinct right-to-left shunt (table). These findings were consistent with chemical pneumonitis. A definite infiltrate in the right lower lobe was first seen only on the third day after exposure.

The patient recovered uneventfully after treatment with parenteral penicillin and was discharged nine days after admission. Pulmonary function returned to normal.[1]

Comment

Mixing solutions of ammonia and sodium hypochlorite produces acrid monochloramine (NH_2Cl) and dichloramine ($NHCl_2$) fumes.[2] On contact with mucosa chloramines decompose to ammonia and hypochlorous acid. The latter combines with moisture, forming hydrochloric acid and liberating nascent oxygen, which is a potent oxidising agent causing most of the cellular injury. Corrosive effects of ammonia and hydrochloric acid also contribute to chloramine-induced respiratory tract damage.

There are no reported cases of serious injury from inhaling chloramine fumes, transient cough, dyspnoea, nausea, and vomiting being described.[3 4] Hence inhaling chloramine fumes is considered less hazardous than exposure to other lung irritants, as expressly stated in recent reference texts.[2 5]

The extent of injury from noxious gases depends on the concentration of inhaled gas as well as the duration of exposure. Chloramine concentrations attained by mixing small or moderate amounts of cleaning agents are probably too low to cause severe injury. This might explain the absence of serious sequelae to chloramine inhalation in other reports. Our case shows that when copious amounts of ammonia and hypochlorite are combined the extent of the bronchoalveolar injury may be considerable. In this respect chloramine is probably no less dangerous than chlorine.

An important observation in our patient was the delayed radiographic appearance of pneumonitis. Pulmonary function tests were more helpful in diagnosing this complication, being commensurate with the clinical findings early in the course. Chloramine exposure from mixing household ammonia with cleaning agents containing hypochlorite is a fairly common domestic accident. The hazards of this poisoning should not be underestimated.

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Results of pulmonary function tests after inhalation of chloramine fumes

Time after exposure	Pulmonary function values (as % of predicted normal[1])								Oxygen pressure/carbon dioxide pressure (kPa)		
	Vital capacity	Residual volume	Functional residual capacity	Total lung capacity	Residual volume/total lung capacity	Forced expiratory volume in 1 s	$Rate_{50}$	$Rate_{25}$	Fractional inspired oxygen: ±50%	100%	21%
48 hours	46	137	85	66	46	42	36	29	6·8/3·6	19·6/4·1	
9 days	93	122	106	99	27	105	100	100			13·0/4·9

Conversion: SI to traditional units: 1 kPa ≈ 7·5 mm Hg.